IN THE UNITED STATES DISTRICT COURT
FOR THE WESTERN DISTRICT OF TEXAS
SAN ANTONIO DIVISION

COL. WILLIAM R. HOGANS III, D.D.S., and GRACE V. HOGANS, MICHAEL D.R. HOGANS, BYRON P. HOGANS, ELLIOTT B. HOGANS, ADRIENNE D. HOGANS, and WILLIAM R. HOGANS IV,	§	
Plaintiffs,	§	
VS.	§	CIVIL ACTION NO. SA-03-CA-439-FB
UNITED STATES OF AMERICA,	§	
Defendant.	§	

OPINION AND JUDGMENT OF THE COURT

Plaintiff Grace Hogans is the matriarch of a wonderful American family. She has loaned her husband and three sons to military training and service. Mrs. Hogans is now, at age 57, bound to a wheelchair and dependent on others for daily sustenance and care. She alleges her condition is the result of a failure by military doctors to use proper diagnostic tools in 1990 to find a tumor, an acoustic neuroma, which she alleges grew to a size that caused a debilitating stroke in 2000. Though she made good recovery, the tumor had to be surgically debulked in 2001, after which her condition worsened to its present state. She asserts if the tumor had been properly diagnosed and treated in 1990 in its smaller size, her damages would not have occurred. Her medical records, some of her treating physicians, and expert opinions support her assertions.

Defendant United States of America contends Mrs. Hogans' stroke and condition are a result of two decades of uncontrolled hypertension, diabetes and obesity. Her medical records and defense expert opinions support this allegation.

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Defendant does not challenge the factual or legal sufficiency of plaintiffs' evidence. Plaintiffs do not challenge the factual or legal sufficiency of defendant's evidence.

This chronology of events appears to be undisputed: On January 25, 1990, plaintiff Grace Hogans had an appointment at Malcolm Grow USAF Medical Center ENT Clinic, located at Andrews Air Force Base in Maryland, and complained of sudden onset unilateral hearing loss in her right ear that had been present since November 13, 1989. She complained of dizziness, imbalance, and a feeling of fullness in her right ear. An audiogram revealed Mrs. Hogans had moderate to severe sensineural hearing loss in her right ear. A computed tomograph (CT) scan was performed on February 22, 1990, and was read as normal.

On December 5, 1991, Mrs. Hogans went to Winn Army Community Hospital, located at Fort Stewart, Georgia, complaining of twitching in her right eye and the right side of her lip. No imaging studies were ordered. She returned on July 13, 1992, complaining of numbness in the right side of her face. Eleven days later on July 14, 1992, Mrs. Hogans again returned to Winn Army Community Hospital complaining of numbness in the right side of her face, light-headedness for the past month, loss of hearing in her right ear and loss of balance. A CT scan was ordered and again interpreted as normal. She returned twice more in August of 1992 with similar complaints, but no magnetic resonage image (MRI) was performed. Despite the revelation, via audiogram performed on March 9, 1993, Mrs. Hogans had profound right ear sensineural hearing loss since the previous audiogram in 1990, no imaging studies were ordered. In fact, an MRI was not performed until Mrs. Hogans suffered a pontine stroke on December 20, 2000, ten years after symptoms first occurred.

Plaintiffs contend the symptoms and signs Mrs. Hogans manifested in 1989, 1990, 1991, 1992, and 1993 were classic signs and symptoms of an acoustic neuroma, and the standard of care

at that time required the government healthcare providers to perform an MRI of Mrs. Hogans' brain to rule out or diagnose an acoustic neuroma. This contention was undisputed at trial because nationally renowned medical witnesses for plaintiffs and defendant agreed the standard of care in Mrs. Hogans' case was breached–an MRI should have been ordered. Moreover, there is no question Mrs. Hogans' quality of living and the lives of her family have been irrevocably diminished. The only contested issue is what cause or causes came together to create her condition.

Both sides presented highly qualified experts to support their causation theory. Neither side filed Daubert challenges. Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579 (1979). The testimony and credentials of these experts are in synopsis form in Appendix A. Defendant did file a motion in limine to limit the testimony of treating physicians.[1] That motion (docket #33) is DENIED. Accordingly, the military treating physicians' testimony is summarized in Appendix B. Following the stroke, the United States military referred Mrs. Hogans to civilian specialist doctors at Harborview Medical Center. Their opinions are set forth in Appendix C.

The Court must break the factual impasse on causation in the context of after-the-fact experts who respectfully disagree with each other. Indeed, Dr. Swann for plaintiffs and Dr. McGrail for defendant are close friends and colleagues but come to diametrically opposite conclusions about the

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[1] Defendant asserts during discovery, plaintiffs took the depositions of several of Mrs. Hogans' treating physicians who were active duty members of the United States military at the time of treatment. These witnesses were asked not only about their care and treatment of Mrs. Hogans but also asked questions reserved for expert testimony. Relying on Young v. United States, 181 F.R.D. 344, 346 (W.D. Tex. 1997), which held that treating physicians generally must be considered as ordinary fact witnesses and should not be considered an expert witness unless specifically retained to develop an expert opinion, defendant requested the Court to prohibit plaintiffs from attempting to elicit such testimony at trial and limit the treating physicians' testimony to that which they have through personal knowledge. In response, plaintiffs state the testimony of the treating physicians will relate directly to the examination, treatment and diagnosis of Mrs. Hogans and will, therefore, be admissible. Based on the arguments of the parties and the testimony presented during trial, the Court finds the motion should be denied.

cause of Mrs. Hogans' damages. Dr. Swann believes the acoustic neuroma caused the stroke while Dr. McGrail believes Mrs. Hogans suffered an occlusion of an artery caused by atherosclerotic disease resulting from hypertension and diabetes.

Although the case was tried in Texas, the parties agree the alleged negligent acts occurred in Maryland and Georgia. Following trial, the Court required the parties to submit additional briefing concerning which state's law was to be applied.[2] The parties agreed that because the alleged negligent acts occurred in two states, this Court must apply the law of the state where the last significant negligent act or omission occurred, i.e. Georgia. (Choice of Law Briefs, docket numbers 49 and 52). The parties also agreed Georgia applies the rule generally known as *lex loci delicti* or the "place of the wrong" approach. Id. In the application of this rule, the parties agree that because Mrs. Hogans suffered her stroke in the state of Washington, this Court must apply Washington law in determining liability and damages. Id. With respect to damages, both parties agree Washington applies a pure form of comparative negligence which allows a plaintiff to recover damages even if his or her fault is greater than that of the negligent defendant. Id. However, as previously mentioned, the parties do not agree on causation.

In Washington, a plaintiff alleging medical negligence is by statute required to prove two elements:

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[2] Defendant filed Defendant's Choice of Law Brief and Motion to Strike Section I of Plaintiffs' Choice of Law Brief (docket #52). Defendant asks the Court to strike Section I of Plaintiff's brief entitled "The Court Need Not Reach the Question of Mrs. Hogans' Alleged Negligence" because defendant contends plaintiffs are merely arguing their case rather than responding to this Court's order and because this section is "unresponsive, irrelevant, redundant, immaterial, and completely misstates the evidence." The Court finds defendant's motion has merit and should be granted. Accordingly, IT IS HEREBY ORDERED that the Motion to Strike Section I of Plaintiffs' Choice of Law Brief (docket #52) is GRANTED.

> (1) The health care provider failed to exercise that degree of care, skill, and learning expected of a reasonably prudent health care provider at that time in the profession or class to which he belongs, in the state of Washington, acting in the same or similar circumstances;
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> (2) Such failure was a proximate cause of the injury complained of.

Morton v. McFall, 115 P.3d 1023, 1027 (Ct. Apps. Wash. 2005). In contrast to Texas law under which the elements of proximate cause are foreseeability and cause in fact, Washington law recognizes cause in fact and legal causation as the elements. Compare Canion v. United States, No. EP-03-CA-0347-FM, 2005 WL 1514045 at *6 (W.D. Tex. June 21, 2005) ("There are two elements of proximate cause under Texas law: forseeability and cause in fact."), with Christen v. Lee, 113 Wash.2d 479, 507, 780 P.2d 1307, 1321 (Wash. 1989) ("The doctrine of proximate cause in Washington entails the two elements of cause in fact and legal causation."). An en banc Washington Supreme Court described the elements as follows:

> "Cause in fact" refers to the actual, "but for," cause of the injury, i.e., "but for" the defendant's actions the plaintiff would not be injured. Establishing cause in fact involves a determination of what actually occurred and is generally left to the jury. Unlike factual causation, which is based on a physical connection between an act and an injury, legal cause is grounded in policy determinations as to how far the consequences of a defendant's acts should extend. Thus, where the facts are not in dispute, legal causation is for the court to decide as a matter of law.
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> The focus in the legal causation analysis is whether, as a matter of policy, the connection between the ultimate result and the act of the defendant is too remote or insubstantial to impose liability. A determination of legal liability will depend upon "'mixed considerations of logic, common sense, justice, policy, and precedent.'"

Schooley v. Pinch's Deli Market, 134 Wash.2d 468, 478-79, 951 P.2d 749, 754 (Wash. 1998). With respect to foreseeability, Washington courts have found it improper to inject this issue into the definition of proximate cause. As one court in remanding a case to a trial court who had determined a reasonable man would not have foreseen the incident and therefore proximate cause had not been established, explained:

> The better considered authorities do not regard foreseeability as the handmaiden of proximate cause. To connect them leads to too many false premises and confusing conclusions. Foreseeability is, rather, one of the elements of negligence; it is more appropriately attached to the issues of whether defendant owed plaintiff a duty, and, if so, whether the duty imposed by the risk embraces that conduct which resulted in injury to plaintiff. The hazard that brought about or assisted in bringing about the result must be among the hazards to be perceived reasonably, and with respect to which defendant's conduct was negligence. In Fleming v. Seattle, 45 Wash. 2d 477, 275, P.2d 904 (1954), the case was submitted to the jury. On appeal this court said: "The actual harm sustained by decedent fell within this general field of danger."
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> It is the misuse of foreseeability–that is, discussion of the improbable nature of the accident in relation to proximate cause–that led the trial judge, in the instant case, to conclude that the challenge should be sustained.
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> It is not, however, the unusualness of the act that resulted in injury to plaintiff that is the test of foreseeability, but whether the result of the act is within the ambit of the hazards covered by the duty imposed upon defendant.
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> We approved this theory in McLeon v. Grant County School Dist., 42 Wash.2d 316, 255 P.2d 360 (1953), in which this court said:
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> > Whether foreseeability is being considered from the standpoint of negligence or proximate cause, the pertinent inquiry is not whether the actual harm was of a particular kind which was expectable. Rather, the question is whether the actual harm fell within a general field of danger which should have been anticipated. This thought is further developed in the following statement by Professor Harper, which we quoted with approval in the Berglund case:
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> > The courts are perfectly accurate in declaring that there can be no liability where the harm is unforeseeable, if "foreseeability" refers to the general type of harm sustained. It is literally true that there is no liability for damage that falls entirely outside the general threat of harm which made the conduct of the actor negligent. The sequence of events, of course, need not be foreseeable. The manner in which the risk culminates in harm may be unusual, improbable and highly unexpectable, from the point of view of the actor at the time of his conduct. And yet, if the harm suffered falls within the general danger area, there may be liability, provided other requisites of legal causation are present.

Rikstad v. Holmberg, 76 Wash.2d 265, 269 456 P.2d 355, 358 (1969). "Reasonable foreseeability" is seen as a "controlling factor in determining the existence of negligence" but not as an element of

proximate cause. Washington v. Giedd, 43 Wash. App. 787, 719 P.2d 946, 948-49 (Wash. App.--1986). As explained in the comment section to the Washington Pattern Jury Instructions:

> The chief value of the "foreseeability" formula is in those cases wherein intervening or superseding forces have come into active operation at a time subsequent to the defendant's conduct. . . . The definition of "proximate cause" should not be further complicated by an attempt to provide for intervening or superseding negligence. This is the proper subject for a separate instruction (e.g., WPI 12.05, Negligence–Intervening Cause.)

Giedd, 719 P.2d at 949.

Here, the evidence supports two distinct theories as the cause in fact of Mrs. Hogans' stroke: the acoustic neuroma or uncontrolled hypertension and diabetes. Although plaintiffs' experts could point to no medical literature in support of their theory, plaintiffs had, in addition to their experts' opinions, the medical records from the physicians who treated Mrs. Hogans at the time of her stroke and who seemed to agree the stroke was caused by the acoustic neuroma. Interestingly, defendant chose not to depose these civilian treating physicians to whom defendant sent Mrs. Hogans. There is therefore no evidence these physicians have changed their opinion. Consequently, the defendant's own contracted doctors support plaintiff's case.

Plaintiffs' theory appears to be based on the location of the acoustic neuroma and the location of the stroke as indicated by the MRI performed following Mrs. Hogans' stroke. Conversely, defendants' experts had medical literature to support their theory that hypertension and diabetes are risk factors for stroke but acknowledged their were no tests to show the existence of the occlusion that caused the stroke here. Defendant's theory is the proximity of the acoustic neuroma to the location of the stroke was merely coincidence because strokes commonly occur in the brain stem as well as in other parts of the brain especially by persons having the same risk factors as Mrs. Hogans.

Moreover, subsequent MRIs revealed, in their opinions, that swelling in the first MRI caused by the stroke made the proximity of the two appear closer than shown in later MRIs.

No doubt the medical debate as to the "cause" of Mrs. Hogans' stroke will go on for years and no one will ever really know what caused the stroke on December 20, 2000. However, because the medical experts cannot agree, this Court must decide based on the evidence presented. The Court finds plaintiffs and defendant have proven proximate cause in equal proportions.

With respect to damages, the experts, Dr. Don Huddle and Dr. Stan Smith (economists) and Dr. Alex Willingham and Kathleen Kuntz (life care planners), have some disagreement but not as diametrically opposed as that of the medical experts. Their testimony is presented in Appendix D. Plaintiffs' damage experts and counsel conclude and argue plaintiffs' loss is $10,986,604.00.[3] Defendant's damage experts and counsel conclude and argue the loss is approximately $1,925,900 depending upon which life care plan scenario is chosen.[4] This Court finds plaintiffs' damages to be $3.25 million caused equally by defendant's negligence and Mrs. Hogans' other medical conditions.

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[3] The breakdown of the plaintiffs' requested damages is as follows:

1.	Grace Hogans:		
	A.	Future Medical, Attendant Care, etc.	$3,327,432.00
	B.	Loss of Earning Capacity	502,432.00
	C.	Physical Pain and Suffering	500,000.00
	D.	Mental Anguish	500,000.00
	E.	Physical Impairment	1,500,000.00
	F.	Mental Impairment	500,000.00
	G.	Disfigurement	500,000.00
2.	Colonel William Hogans, III:		
	A.	Care Provided to Grace Hogans in the Past	$ 93,060.00
	B.	Loss of Consortium	750,000.00
	C.	Loss of Household Services	34,500.00
3.	Adrienne Hogans		
	A.	Care Provided to Grace Hogans in the Past	$ 279,180.00
	B.	Loss of Consortium	500,000.00
4.	William Hogans, IV, Elliott Hogans, Bryon Hogans and Michael Hogans the sum of $500,000 each for Loss of Consortium		

[4] Dr. Smith opined Mrs. Hogans' earnings loss, past and future, to be $195,707. The cost of Mrs. Hogans' future life care varied depending upon various scenarios. The estimate for scenario 1 was $1,730,193; scenario 2 - $811,919, and scenario 3 - $787,402.

Applying Washington state comparative liability concepts, IT IS HEREBY ORDERED that judgment shall be entered in favor of the plaintiffs against the United States of America in the total amount of $1.625 million apportioned as follows:

1. Grace V. Hogans shall recover the sum of ONE MILLION AND NO/100 DOLLARS ($1,000,000.00).

2. Colonel William R. Hogans, III shall recover the sum of TWO HUNDRED FIFTY THOUSAND AND NO/100 DOLLARS ($250,000.00).

3. Michael D. R. Hogans shall recover the sum of SEVENTY-FIVE THOUSAND AND NO/100 DOLLARS ($75,000.00).

4. Elliott B. Hogans shall recover the sum of SEVENTY-FIVE THOUSAND AND NO/100 DOLLARS ($75,000.00).

5. Adrienne D. Hogans shall recover the sum of SEVENTY-FIVE THOUSAND AND NO/100 DOLLARS ($75,000.00).

6. William R. Hogans, IV shall recover the sum of SEVENTY-FIVE THOUSAND AND NO/100 DOLLARS ($75,000.00).

IT IS FURTHER ORDERED that plaintiffs shall recover their costs of court from the defendant.

Post-judgment interest shall accrue and be payable on all of the above amounts at the maximum rate allowed by law from the date the Judgment is filed with the appropriate government agency until said Judgment is paid.

It is so ORDERED.

SIGNED this 30th day of September, 2005.

FRED BIERY
UNITED STATES DISTRICT JUDGE

APPENDIX "A"

Plaintiffs' Experts:

1. Dr. J. Neal Rutledge–Board certified in radiology and a practicing neuroradiologist since 1997. Medical Director Neuroradiology at Seton Health Care Network and Chairman of Stroke Committee at the Brain and Spine Center. He reviewed the MRIs and CT scans of Mrs. Hogans

   He believes, based on reasonable degree of medical probability, the U.S. government healthcare providers fell below the standard of care in February of 1990 when they ordered a CT scan and not an MRI. He believes an MRI should have been ordered because it would have been able to visualize the acoustic neuroma in the internal auditory canal. He also believes the United States government healthcare providers fell below the standard of care in July of 1992 when they ordered a CT scan. The standard of care would have been to order an MRI.

   He believes if the acoustic neuroma could have been identified when it was within the canal, it could have been surgically removed without causing the subsequent stroke.

   His opinion based on reasonable degree of medical certainty is the cause of Mrs. Hogan's stroke—the mechanical compression on the pons, occluding the vessels.

   He explained Mrs. Hogans' injury to the pons in two parts as follows: It's cutting off the blood supply, but the question is or the question that has come up is where the blood supply cutoff. And that's why people are arguing about the mechanism. So what we did is we took this soft spongy ball and we put concentric circles in it so you can see where the pressure is actually formed versus we have this golf ball which is hard. When you see, if this represents the pons, and the tumor is hard when you see it press against the pons you can see that it sort of in this area in here, where the maximum where the maximum pressure occurs. It presses on both the arteries and veins and when you don't get blood in or you don't get blood out. That's where the tissue dies.

   Exhibit 115 is an MRI taken on December 21, 2000. Dr. Rutledge explained the MRI scan was designed to show water inside the cell where there's mechanical breakdown. So the white is sort of where there's cellular breakdown and the rest is sort of gray. So right there is the tumor as they outlined before. This is the area where there's the white is the cellular breakdown or the ischemia, the infarct and actually that whole area the white and this area over here, that's the pons. It's been just like that softball it's been squished by the tumor which is actually the schwanoma.

   Dr. Rutledge acknowledged he has not published any articles relating to the diagnosis or treatment of acoustic neuromas.

He testified he has never seen an acoustic neuroma cause a stroke because usually they are very small and not pressing on the brainstem and so if the tumor does not get that large, it cannot act in this way. Although he has never seen an acoustic neuroma cause a stroke, he believes that because of the unusual size, because it was allowed to get as big as it was, it's the reason why it caused the stroke and also because of its positioning; because of MRI scanning he noted doctors just do not see tumors this large anymore.

Agreed that at the time of the December 20, 2000 stroke, Mrs. Hogans had an old infarct. Also agrees Mrs. Hogans had more than five significant risk factors for some type of stroke.

When asked if there is any literature that states an acoustic neuroma is an independent risk factor for a stroke, he replied that it is "in that one of the risk factors of acoustic neuroma is death and the mechanism of death is stroke." The literature may not say stroke what it says is death and "you have to ask yourself what causes the death? Well, it's the compression of the tumor against the brainstem so I think it's I think it's an argument of semantics." No literature where patient has suffered a stroke as a result of an acoustic neuroma because nowadays the tumors don't get as large. He is not aware of any literature that says acoustic neuromas cause strokes.

He believes the acoustic neuroma pressed against the outflow veins and did not let the blood get out of the pons.

His opinion is the mechanism of the injury in this case was due to a vein obstruction. He disagreed that the stroke was caused by obstruction of perforating arteries and not veins. When asked, "you understand that both the plaintiffs' neurosurgeons in the case when asked about the mechanism of injury testified that they believed it was the perforating arteries, not the veins?" He replied he did not disagree with them just the way they described it.

He believes the person in the best position to determine the mechanism of the injury is the neuroradiologist.

He agrees there is a variance among MRI machines and their strengths. He agrees that Mrs. Hogan's facial paralysis is due to complications or due to surgery she underwent at Harborview Medical Center in May of 2002.

He has never testified as an expert witness in a case involving an acoustic neuroma.

When asked if there could have been more than one proximate cause of the event, he responded: "I mean you can never say never. You know in regards of the arguments about diabetes and hypertension but it's just it's extremely unlikely." He believes this stroke was from the tumor pressing on the brainstem.

2. Dr. Gerald Paul Larson – Board Certified Otolaryngologist.

Otolaryngologists are medical and surgical specialists for the head and neck; take care of diseases of the ear, nose, sinus, throat, larynx and tumors of the neck.

He has seen an acoustic neuroma cause a stroke. He explained when he was a resident in surgery he did a neurosurgical residency and spent time in the neurosurgery department and at that time diagnosed tumors of the brain with arteriograms. "And basically, it was just the displacement of these arteries, is how we determined a mass that was in the intercranial cavity. It displaced the known major vessels." During that experience he knew of two people who presented with tumors or with a stroke who were subsequently diagnosed with the acoustic neuroma as the etiology of the stroke. He has not seen a patient suffer a stroke from an acoustic neuroma since the advent of the MRI. The reason for this he explained was because doctors could even further investigate these tumors and find them within the internal auditory canal; tumors millimeters in size could be found.

A otolaryngologist is an ear, nose and throat doctor.

When asked about a breach of the standard of care in this case, he responded: "Actually, I think the ordering of the CT scan was not the most preferable at the time but it was compounded also by the fact that they may have not had an MRI and if they would use that as their preliminary examination but then on the report the radiologist actually reiterated the fact that the MRI is the most sensitive."

The United States government fell below the standard of care in 1992 when Dr. Sommer ordered a CT and not an MRI. In 1992, the MRI was widely available.

Dr. Larson was also asked if he had an opinion about whether the breaches of the standard of care caused the injuries and damages sustained by Grace Hogans and the Hogans family. Defendant objected because Dr. Larson is not a neurosurgeon and did not deal with anything intracranially and that is where they are alleging the damages occurred. The Court noted that looking at Dr. Larson's report on the second page Dr. Larson states "'The tumor ultimately became so large that it grew adjacent to the pons and caused an infarction by direct compression,' which, of course, we've heard that language before. 'Ms. Hogans now lives with a severe result of the brain stem stroke.' So Doctor, do you – your main area of expertise, of course, is the ear, nose, throat and so forth. And the pons and all of that is the brain and which is the basis of Mr. Dietrichs' objection. Given your area of expertise – and there, of course, have been no objections as far as you testifying concerning your opinion as to what technology should have been used to find this acoustic neuroma. But now it seems that we're going the next step. What is the basis of your expertise to get then into that next step?"

Dr. Larson responded: "I would – I would say, sir, that my knowledge now would be that of a just a general medical doctor, anatomy and physiology that I learned in medical school. I do have a little bit more because of my residency training." The Court: "Right. And then, of course, you did testify of having at least recalled seeing strokes resulting from these back before the MRI technology was available." Dr. Larson: "Yes sir."

The Court overruled the objection and allowed Dr. Larson to answer the question. In Dr. Larson's opinion, "the continued neglect of the diagnoses of the acoustic neuroma allowed it to grow to a considerable size and cause the defects that she suffers from." His opinion as to her stroke: "It's the compression of the brain substance by the acoustic neuroma causing loss of nerve function."

Dr. Larson agreed Mrs. Hogans would have had the stroke even if she had never had diabetes, blood pressure, weight problems, so forth. He also stated his opinion was her hypertension had no relevance to her stroke. He explained he has had the opportunity to see the data that was accumulated since his deposition was taken and "they had studies that were done actually using Doppler studies of the blood flow. And these were all considered to be normal. And I think we had some other effects of some diplopia and things that were later after the stroke that actually got better after her surgery. And that leads me to conclude that if these were caused by chronic metabolic changes of the arteries themselves by hypertension and diabetes, that they wouldn't resolve. So I think this is more of a logical assumption, with my medical training.

In his opinion, based on reasonable degree of medical certainty, is that if Grace Hogan's acoustic neuroma had been diagnosed and treated appropriately she would have suffered the stroke.

He concludes based on the second CT scan that was done in 1992, that the tumor was still inside the ear canal at that time.

Dr. Larson does not operate on acoustic neuromas or strokes and it is not in his practice to diagnose or treat strokes. He also does not read MRIs of the brain.

Dr. Larson stated that ear wax was the most common cause of unilateral hearing loss.

In his 20 years of private practice he has discovered an acoustic neuroma in about 5 patients.

Of all the physicians testifying, Dr. Larson is the only one testifying that the tumor is a large neuroma. By and large the treatment for an acoustic neuroma is surgery. He disagreed with Dr. Scholl and Dr. Swann that a watchful waiting is an appropriate course of action.

Mrs. Hogans' hearing loss was not salvageable in either 1990 or 1992.

He was unaware that Mrs. Hogans had had other strokes. He did not read in the medical records where she was diagnosed on MRI at Harborview with two prior strokes. He said that information would have had an affect on his opinion.

3. Dr. Karl Swann - board certified neurosurgeon.

As part of his practice he treats patients diagnosed with acoustic neuromas.

Based on a reasonable degree of medical certainty he believes the United States healthcare provider employees' care fell below the standard of care in 1990 and 1992 by failing to obtain a brain MRI scan. He opinion, based on a reasonable degree of medical certainty, is that these breaches caused the injuries and damages sustained by Mrs. Hogans. His opinion:

> Well, the failure to diagnose the acoustic neuroma that was almost certainly present at the time allowed it to expand to a point that caused her harm.

Had the healthcare employees performed an MRI in 1990, in his opinion the status of the acoustic neuroma was probably what is referred to as an intercanalicular acoustic neuroma, much smaller than when it was ultimately discovered in 2000.

He explained what the difference would have been between an MRI taken in 1990 and the one actually taken in December of 2000 as follows:

> Well, this is, as I said, a rendition of an intercanalicular acoustic neuroma. And what happens is the tumors – these tumors start in this general region and then follow the path of least resistance out the – from the canal, internal auditory canal, and up against the brain stem in the case of Mrs. Hogans. This is the pons. And then this is the cerebellum. And obviously, the tumor is much – is much bigger on the right then the rendition on the left.

He explained the effect the tumor was having on the pons as follows: Well, pressure. And that's – that's what we see here with the distortion of the pons. This is what's called the fourth ventricle which is the – part of the spinal fluid passageway inside the central nervous system." And the tumor which is here, the white, is pressing on the brain stem and actually pushing the fourth ventricle away from it and distorting the normal anatomy.

Had the acoustic neuroma been diagnosed in February of 1990, Mrs. Hogans would have had three treatment options: first option to follow the tumor with serial scanning; second option would be radio surgery such as gamma knife and then third option would be surgical removal.

His opinion based on a reasonable degree of medical certainty concerning Mrs. Hogans' damages had the acoustic neuroma been diagnosed and surgically removed in 1990 was explained: Well, with gamma knife, a successful gamma knife the tumor would remain exactly as it is it doesn't go away and that's just to clarify for the Court that zapping it doesn't make it go away. It just sits there so she would retain the tumor in its state. Operating on it, probably she would have lost whatever remaining hearing she had but probably especially with a tumor that size you have a very low risk of injury of the facial nerve so just minus her hearing and really in both instances either the gamma knife or the operative.

Serial MRIs is an option because the tumors are relatively slow growing. Occasionally you run into somebody where the tumor does grow real slowly and it allows the patient some time to think about it and let the reality of it all sink in, but the point is to try to catch a tumor before it gets too big because the larger the tumor, the greater the risk of operating.

He agreed it is also important to catch the tumor before the patient starts developing more symptoms and it becomes more symptomatic for the patient.

His opinion on the difference in surgery in 1990 versus the surgery she had in 2000 was as follows: Well, first of all just in time, in the sense of time, an operation on a small tumor such as - such as this one, you're talking maybe at most a couple of hours as opposed to the larger tumor probably four to six hours depending on what you're doing debulking or trying to remove it. The surgical risks are different as well. He mentioned the smaller tumor has a lower risk of injury.

"The goal of - for treatment of any benign tumor is to try to remove the whole thing if you can. The debulking procedure is to get inside the tumor and take out the core, not touching the - what's called the capsule or the outer portion of the tumor." In Mrs. Hogan's case approximately 80% of the tumor was removed.

His opinion as to why the entire tumor was not removed: "Well, they felt, I think, that the tumor was adherent to the brain stem and that they did not want to cause any further injury, further stroke to Mrs. Hogans."

His opinion as to cause of Mrs. Hogans' stroke: "It was the pressure caused by the acoustic neuroma on the - on the brain stem affecting the blood supply to the brain stem." It is his opinion based on a reasonable degree of medical probability that the tumor compressed the pons causing the stroke.

His explanation of the MRI done on Mrs. Hogans the day after her stroke on December 21 at Harborview Medical Center: it means that the neuroradiologist who's reading the MRI scan has looked at the study and says that there is an abnormality in the pons as the person says they're adjacent to the CP angle mass which is where the her tumor was,

having marked mass effect means that it's compressing it, pushing on it, distorting it, and then this foci of hyper - intense diffusion signal consistent with acute infarct means that the radiologist sees a stroke basically inside the pons and then the radiologist goes on to make a statement why – what the most likely reason for that is and says that it is secondary to or because of occlusion or pressure on and narrowing of actually occlusion means plugging up. Translation to a lay person – the tumor caused the stroke.

Exhibit 120 shows the acoustic neuroma right adjacent or touching the stroke.

In taking issue with Dr. Chalela's opinion, Dr. Swann said he thinks the pressure from the tumor caused the stroke. The testimony by Dr. Chalela assumes, in Dr. Swann's opinion, a couple of things and ignores one important thing: First of all, it assumes that Mrs. Hogans had textbook anatomy. This, what we're looking at here is a drawing, an artist's rendition of something that's used to basically teach students. And so it represents an average or somebody's idea of what an average is. Most people don't necessarily have average anatomy. In fact, he noticed something on the MRI scan, basilar artery was dead mid line in the case of Ms. Hogans right there this little circular dark area is her basilar artery and this is the mid line right here. So even in her, this is a good illustration of how people just don't look like textbooks, that her artery is not dead center and so the assumption of a neurologist, that this particular patient has the anatomy that's precisely like that drawing which usually not the case.

The second thing [Dr. Chalela's opinion] ignores is the evidence that or it assumes this is the neurologists testimony that there is a totally other process going on in Mrs. Hogans' brain, namely some type of process that caused this stroke and there's no evidence in the medical records that she does have large plaques in her basilar artery or sources of emboli coming from somewhere else. So it assumes basically perfect textbook anatomy which most people don't have, it assumes a totally separate pathologic process to cause a stroke, which there's no evidence of. And then thirdly it ignores the big tumor that's pressing on the – on the brain stem right adjacent to where the problem is.

He thinks he has seen an acoustic neuroma cause a stroke. It was during his training, 1979 through 1986. But he has not seen one since.

It is not surprising that no articles have been written in this area because probably back at least during his training they did not do MRIs; the advent of MRI was toward the end of his training so most of what he was dealing with was CT scans which were not as sensitive as an MRI. People seem to have MRI scans earlier these days and so the tumors do not get quite this big. So there is not a large population of patients out there to have strokes as a result of the tumor compression. He agree that since the advent of MRIs in the late 1980s to early 1990s, acoustic neuromas are diagnosed long before they get to the point where they are compressing the pons and occluding vessels.

He agrees with the general proposition that if a patient has diabetes and hypertension they are at a slightly increased risk for having a stroke, but he did not find any evidence in the records that diabetes or hypertension caused Mrs. Hogans' stroke. He disagrees that Mrs. Hogans was at an increased risk for strokes because she allegedly had had two prior strokes. Nor does he agree if a patient has lacunes that that patient is at an increased risk for strokes.

He believes the tumor was allowed to grow unchecked for ten years causing compression of the vessels in the pons causing their occlusion and causing the stroke.

His interpretation of the report by the doctors on the day of the stroke: the radiologist concluded that "there's a tumor that's pressing on the brain. And then she sees something else abnormal on the scan which is the stroke, of course, and that that's – she's drawing the conclusion that the two are related.

The attending neuroradiologist at Harborview Medical Center basically concluded, according to Dr. Swann that "[t]he tumor is having marked mass effect, which means it's pressing on it and distorting it. And there's the hyper-intense diffusion signal consistent with the infarct, which means stroke. And then this radiologist is concluding that the stroke is most likely secondary to, or because of, occlusion of the perforating vessels which are the small, little blood vessels that come off the outside of the brain stem by this mass." An attending neuroradiologist is a fully trained probably board certified neuroradiologist not a resident in training.

His translation of the records from Harborview is that the tumor caused the stroke. That this staff neuroradiologist has evaluated the situation clinically, has presumably examined the radiographic studies and concludes that the source of the stroke is pressure on the brain stem by the tumor occluding or compressing the blood vessels, causing the stroke."

Six days after the stroke, the attending rehabilitation physician wrote that Mrs. Hogan was found to have compression of the right side of the pons and an infarct. Although this comment isn't quite as strong as the others that the tumor caused the stroke, Dr. Swann states it indicates a relation.

Eight days after the stroke, the short version of the medical record is interpreted as basically similar to the last. The records generated on January 18 and November 19, 2001, agree that the tumor caused the stroke. Also the record of Dr. Chan Hwang on May 26 of 2001 also agrees.

There is no evidence the stroke was caused by blood vessels being burst by pressure in the brain or bleeding. A echocardiagram was done in the days following her stroke to look for a clot and it was normal. The tests following the stroke showed no clots, no narrowing and no abnormal blood flow. They also did an angiogram of the basilar artery for 20 minutes and did not find any source of stroke any source of emboli.

His opinion based on a reasonable degree of medical probability is that the pressure on the pons, distortion of the pons, and then distortion of the 6th nerve caused the problem with double vision and left lateral gaze and it was not the result of diabetes and hypertension.

He explained if a person has diabetes and hypertension, the eyes are typically one of the first places if you are going to have any narrowing or clotting as a direct result of diabetes and hypertension. Mrs. Hogans had her eyes examined 2 months before the stroke and the vessels in her eyes were normal. The vessels in the eyes are very similar to the vessels in the pons that were compressed by the tumor.

He agrees that throughout the records there is "unanimous agreement that the tumor squashed the pons and put pressure on the pons and that is what caused the stroke." And this is his opinion based on reasonable degree of medical certainty. He also thinks the stroke was the result of the tumor putting pressure and not the result of diabetes or hypertension.

He is aware that Dr. Yu (plaintiff's expert neurosurgery witness), Dr. Powers (defendant's neurosurgery expert) and Dr. Chalela (defendant's expert in stroke neurology) had never seen a case where an acoustic neuroma caused a stroke. Nor had any of his colleagues he had spoken with ever seen an acoustic neuroma cause a stroke. He also acknowledges that the medical literature reports no case of an acoustic neuroma ever causing a stroke.

He thinks at least once, maybe more than once, has he seen an acoustic neuroma cause a stroke—if memory serves him right it was during his residency, i.e. a long time ago. This memory is a "vague foggy recollection of something that might have occurred." He stated he could not give the name of the patient or the exact circumstances, "but I think it probably did. I just - but it is a vague and foggy memory, that's right."

He also agreed that he had testified in his deposition that his colleagues told him they had seen strokes occur in patients after undergoing surgery for the removal of an acoustic neuroma which was the result of an surgical complication and not due to the acoustic neuroma itself. He agreed that in site of the fact that there is no medical literature, scientific literature to support acoustic neuroma causing a stroke it is still his testimony that that is what occurred in this case.

He agreed it would be fair to say that because the acoustic neuroma is in the same area as the stroke that the acoustic neuroma caused the stroke. That, plus the fact there was no other reason for her to have a stoke in the same area.

He agreed that small vessel ischemic strokes could be caused by high blood pressure. He agreed obesity is "probably" a risk factor for stroke. He agreed diabetes is a significant independent risk factor for stroke and high cholesterol is probably a risk factor. The stroke rate is also higher in African-American women than for Caucasian women. The more severe the hypertension and diabetes the greater the risk of stroke.

He agreed that medical literature states transient ischemic events otherwise known as TIAs are stroke predictors of stroke and the American Heart Association has published material that says a person who has one or more TIAs is almost ten times more likely to have a stroke than someone of the same age and sex who has not.

He remembers that the CT taken at Harborview on December 20, 2000, showed Mrs. Hogans had a lacune. He defined lacunes as basically small strokes, brain tissue loss.

When asked to take all five of the risk factors, diabetes, high cholesterol, previous TIAs, being African-American, hypertension, and place them in one patient, he agreed it increases the risk but was not sure if the increased risk was "exponentially."

He agreed that if you look at the stroke literature and the factors that put you at risk for stroke, you will never find a scintilla of medical literature that states an acoustic neuroma is an independent risk factor for stroke. He agreed there are other tumors that do that, but there is nothing in the literature that describes the foregoing.

Acknowledges that he and Dr. McGrail are friends. Dr. Swann was chief resident when Dr. McGrail was a junior resident. Dr. Swann considers Dr. McGrail a very accomplished neurosurgeon who is chairman at Georgetown University. He is aware that Dr. McGrail testified that "given all of the risk factors for stroke that Mrs. Hogans had, compared to complete absence of medical literature to support an acoustic neuroma ever causing a stroke, it would require an enormous jump to come to the conclusion that the acoustic neuroma caused the stroke in this case."

Although there is no medical literature to support his opinion, Dr. Swann contends the opinions are not always necessarily based on what is in the medical literature. "We can't ignore the medical evidence that our patients. Sometimes we're called upon to make judgments based on the information that we have not necessarily what's out in the literature."

He acknowledged there is no study that shows that acoustic neuromas cause stroke.

He disagrees with Dr. McGrail's opinion that if this case was presented to a peer review for publication it would be turned down because of insufficient evidence because you cannot show that the acoustic neuroma caused the stroke when you have all of these other causative factors that existed in this patient. Dr. Swann thinks this would have been a great case to present, and he disagrees it would have been rejected. Dr. Swann is surprised someone has not published it because it would at the very least have prompted discussion in the literature and perhaps brought out other cases that people were either unsure of or very sure of.

Although risks are associated with any surgery involving an acoustic neuroma, the larger the tumor, the greater the risks for more serious problems.

He agreed Dr. Winn's debulking surgery had some further exacerbating effect on her condition. In fact, her current state resulted after the debulking procedure. This was because she was no longer using a wheelchair prior to surgery, she did not have any facial drooping, facial paralysis and most of her other symptoms with regard to speaking had corrected after the December 2000 stroke.

He agreed with the Court that if the MRI had been done back in 1990 or 1992 when the acoustic neuroma was very small, all risk would not have been eliminated but the risk would have been substantially reduced.

He described the size of Mrs. Hogans' tumor as medium. He also agreed that he would not agree that the reason the tumor caused a stroke in Mrs. Hogans was because it was so big.

He answered "correct" to the statement "Because you as practicing neurosurgeon actually Dr. McGrail has told me his residents participated in surgery on acoustic neuromas that were larger than that this never caused strokes." The literature that talks about surgical removal of acoustic neuromas much larger than Mrs. Hogans' do not reference strokes caused by it. In this situation, he thinks it was the size relative to the vascular anatomy that caused the stroke.

He has nothing to dispute Dr. Yu's testimony that between 1991 and December 20, 2000, Mrs. Hogans did not make any complaints of a progression of her symptoms. He also believed Mrs. Hogans' acoustic neuroma did not grow between December 2000 and May of 2002.

He does not believe the stroke was caused by a venus outflow obstruction but rather caused by a perforating artery or a number of perforating arteries—occlusion of. It was an artery not a vein.

He agreed with Dr. McGrail who testified in his deposition that in reference to the entries in Mrs. Hogans' medical records concerning her stroke caused by the acoustic neuroma, that often times in medical records once something is written it get perpetuated. He later qualified this is being "pretty unlikely."

Dr. Swann never talked with any of the physicians at Harborview.

He agrees that an acoustic neuroma is one of the more common benign brain tumors. He answered "yes" to the following, "And yet we've never seen in acoustic neuroma literature cited as a cause of a stroke?"

He agreed there was no evidence in this case showing diabetes or hypertension caused either a narrowing of the arteries or veins or vessels in the pons or that it caused a clot to lodge in the vessels cutting off the blood supply. He stated there is no evidence in the medical testing that was performed to suggest that diabetes or hypertension caused the stroke in the pons.

His opinion, based on a reasonable degree of medical probability, that what was causing the double vision, the lateral gaze, and the difficulty walking Dr. Winn wrote about was the distortion of the brain stem by the tumor.

Had the tumor been diagnosed and treated in 1990, Ms. Hogans would not had to have undergone the debulking surgery in 2000. If the tumor had been diagnosed and appropriated treated and surgically removed in either 1990, 1991 or 1992, the tumor would "probably not" have compressed on the brain stem and on the pons.

Mrs. Hogans would "probably not" have had any of the injuries as a result of having a surgery in 2002 if the tumor had been appropriately diagnosed and removed 10 years earlier.

His opinion based on reasonable degree of medical probability is that Mrs. Hogans would not have sustained any type of symptoms of compression of her pons had the tumor been appropriately diagnosed and treated in 1990, 1991 or 1992.

Had the tumor been properly diagnosed, none of this would have ever happened. He testified that it is really rare for people with hypertension and diabetes to have brain stem strokes. And given that scenario, you have to figure out why she had a stroke in this particular area. You have the tumor and then you have the other studies that don't show the vascular problem that one would expect to see if the stroke was due to the high blood pressure or the diabetes.

4. Dr. John S. Yu - board certified neurosurgeon. He is the Co-Director of the Comprehensive Brain Tumor Program at the Maxine Dunitz Neurosurgical Institute at Cedars-Sinai Medical Center.

In 1990 and 1992 the standard of care required an MRI to rule out an acoustic neuroma as the cause of Mrs. Hogans' symptoms.

The acoustic neuroma grew uncheck to a size of 2.8 cm x 2.5 cm x 2.2 cm., resulting in the tumor adhering to the pons, placing pressure on the pons, distorting the pons and brainstem, and causing the surgery to debulk the tumor to be more complicated and riskier.

As a direct result of the debulking surgery, Grace Hogans suffered serious, permanent neurological and physical injuries all of which would have been avoided had the

government healthcare providers ordered an MRI in 1990 or 1992 and treat the acoustic neuroma while it was intracanalicular.

Assuming an acoustic neuroma would have been found in 1989, the consideration would have been to follow Mrs. Hogans with serial MRIS every six months to a year depending on the stability of the tumor. It would be pure conjecture to speculate what the size of Mrs. Hogans' tumor would have been if found in 1989.

Hypertension and diabetes would have had very little impact on a decision to surgically remove the acoustic neuroma in 1989 because of Mrs. Hogans' age at the time.

The size of the neuroma changes the risk assessment–increasing the size will increase the risk of the surgery as well as radial surgery. As the tumor gets larger, it encompasses the nerve and envelops the nerve such that it makes the dissection of the tumor away from the nerve more difficult and more fraught with risk.

He associates the tumor with the infarct. Dr. Yu explained: the presence of the tumor immediately adjacent to the area of the infarct. A very likely etiology of the infarct being secondary to the compression of the tumor on blood vessels that supplied the pons.

None of the acoustic neuromas that he has observed, diagnosed or treated has caused a stroke. He has not found literature or a case report or study that linked an acoustic neuroma with a stroke but he has never specifically looked for that. The sheer nature of the proximity of the tumor to the infarct leads him to conclude it was the tumor that was directly causative of the stroke.

It appears the tumor was large and compressing the brain stem and causing significant mass effect upon it. The tumor also encompassed the area where a major vessel exists on the brain stem. Dr. Yu states by putting two and two together, it is unavoidable but to conclude the tumor was the causative reason for the infarct.

Perforators are small arteries that emanate from larger major arteries that perforate or go through the brain substance to provide perfusion or oxygenation to the brain matter.

He never met or examined Mrs. Hogans, and he was not involved with her treatment.

Fair to say the facial weakness and now being confined to a wheelchair were attributable to the debulking surgery and not related to the sequelae from the infarct of 2000. That same outcome was a risk had she had surgery in 1990 but the risks are certainly diminished as the size of the tumor is diminished.

There are no progression of symptoms in the medical records from 1992 to 2000. Although it is possible her tumor did not grow during this time, Dr. Yu opines the more

likely scenario is that having had her problem evaluated twice, she may have assumed all possibilities had been ruled out. Although evidence of symptoms means the tumor is growing, the absence of symptoms does not mean it is not growing. Dr. Yu acknowledges there was a two-year period of time where no growth occurred.

Defendant's Experts:

1. Peter Scholl–board certified Otolaryngologist

He has been practicing since 1986. He could not find any literature showing a correlation between an acoustic neuroma and the type of stroke Mrs. Hogans had.

He could not find any literature in the time frame pre-MRI but after using CT's that indicated that the acoustic neuroma had caused a stroke like the one Mrs. Hogans had. Today, patients having large acoustic neuromas are still found. A large acoustic neuroma is one over 3 centimeters. Mrs. Hogans' acoustic neuroma was around 2.8 by 1.5 which is in the medium class. The reason patients still have medium to large acoustic neuromas today with the available technology is because they don't seek care, they don't have available care or the symptoms can be intermittent. It is possible for a patient to not have symptoms until the acoustic neuroma is in the medium to large range.

None of the patients he has seen who have come in after the advent of the MRI who had medium or large acoustic neuromas have had a stroke like Mrs. Hogans.

Acoustic neuromas can be removed, treated with radiation therapy (gamma knife) or observed and watched for growth. The gamma knife probably did not come into general practice until the mid to late 90s.

When a patient comes in with an acoustic neuroma, he does not advise them of the risks of stroke because he had never seen it nor has he seen it in literature.

He agreed an MRI was clinically indicated in Mrs. Hogans' case. He agreed the government health worker employees fell below the standard of care when they failed to do an MRI in February of 1990. He agreed the probabilities would have been very high to have diagnosed the acoustic neuroma by MRI in 1990. Also agrees the standard of care in 1992 for the government health care workers was to do an MRI in Mrs. Hogans' case based on her signs and symptoms. More likely than not the neuroma would have been diagnosed.

He is not rendering an opinion about the cause of the stroke because it is outside the area of his expertise.

He is aware Dr. Larson, an otolaryngologist, testified the tumor pressing on the pons caused the stroke. In Dr. Scholl's opinion that is not within his area of expertise.

The location of Mrs. Hogan's tumor is a typical location.

2. Dr. Alexandros Powers - board certified neurosurgeon

He does not agree with the theory that the acoustic neuroma pressed on the pons causing mass effect which occluded arteries causing the stroke. He disagrees with the physicians and medical records that state the mostly cause of the stroke was the tumor impinging on the brain stem and possibly interrupting the blood flow.

He disagrees based on subsequent medical data. He explained: we have additional information because we have the chance of seeing further imaging studies; we also have a chance to have in the medical record an evaluation of her recovery following the events. This information was not available to the Harborview physicians. These physicians did not see the subsequent MRI scans, the original MRI scan was December and approximately three months later in March there was a follow-up MRI scan.

He was asked if the physicians at Harborview Medical Center made an error when they said presumably this stroke was caused by the pressure from the tumor. His response: With the qualifier presumably, they were qualifying their statement and I assume that means they didn't know. Ultimately it proves that was not cause that was not the mass effect but at that time they were just saying we don't know and this is a possibility, this is part of our differential, then that is accurate, it can be part of the differential ultimately with work up it proved not to be the case.

He says just to the left of where the stroke took place is the tumor. He does not agree with plaintiff's expert who said the stroke is directly adjacent to the tumor. He explained: Because this is in the pons, but if a tumor was immediately adjacent and it was, for sake of argument, pressure phenomenon, the blood vessels that would be affected are the smaller vessels in the mid portion of the pons and so that stroke adjacent to the mass would be like a lacune, a little lake, just a small – and it would have a circular appearance, as opposed to this wedge-shaped defect. And so this tissue here is being supplied by the artery. And that is not right next to the tumor.

The later MRI shows the Harborview diagnosis inaccurate because this does show this distribution of an arterial supply, unlike what one would expect with the mass effect of a tumor would generate an effect on the small blood vessels in the mid portion of the brain tissue and that would generate a lacune, so the abnormality would be just in this quadrant as opposed to in this distribution as I mentioned which is the arterial supply.

He agrees with Dr. Swann's testimony that a lacune is mini stroke.

He disagreed with plaintiff's ball demonstration/exhibit as follows: Although visually they look like they should, these are different consistencies. And you are taking a hard structure and pushing into a soft structure. Brain tumors for example, acoustic neuromas and the brain itself, there is not that difference in tissue tension and the reason I can tell you that is because I move these things and I take these things out and I feel the brain tissue and I feel these tumors. So the consistency is very close, and this is not – this is inaccurate.

The pons and the tumor are of the same consistency. They would not be pushing the same way that the hard ball would into this soft ball that the plaintiffs provided.

He finds the occlusion coming off of the basilar artery. What caused the occlusion of this vessel is the long history of poorly controlled hypertension and diabetes.

He finds the stroke suffered by Mrs. Hogans is inconsistent with the pressure – mass effect of the acoustic neuroma. Although Dr. Swann testified there was no evidence in the medical records of any microvascular disease as a result of hypertension and diabetes, Dr. Powers states we have the lacune infarcts which are identified on the MRI scans.

Because the location so far away from the pons does not have any impact on his belief that the stroke in the pons was a result of microvascular disease. "Because miscrovascular disease is a continuum. So what is happening is the effects of the hypertension and the diabetes is going to have an effect on vessels of the same size. So the vessels that form these lacunes higher up in the brain have similar anatomy characteristics to the vessels in the pons. So the fact that we see this here is telling us that those vessels are at risk and it would be consistent to say in the continuum of small vessel disease if there was enough to cause an effect in a lacune up high in the front part of the brain, top part of the brain. It will have an effect on the back of the pons."

Dr. Swan testified there are three reasons he believed there is no evidence of hypertension or diabetic microvascular disease: the first is there are two tests for retinopathy that Mrs. Hogans went through, one only two months prior to her stroke and they found no occlusion of microvessels in the eye. Dr. Swann testified that that is – that shows that there was no microvascular disease in the microvessels of the brain. He disagrees because from a structural point of view, the vessels in the eyes do have similar size but it is a completely different physiology. They are in a different area, they are not exposed to the pressure forces that these types of vessels are exposed to so it is a different setting in which those stresses function.

The brain is the only organ where that is a constant positive pressure on the vessels and it is just between high to moderate it is always there. The pressure in the eyes is much

lower. It is not a valid comparison to looking at whether somebody is suffering microvascular disease in the brain to compare the miscrovessels in the brain to those in the eye.

He also reviewed the echocardiogram taken of Mrs. Hogans to support plaintiffs' theory that hypertension did not cause the stroke. Dr. Powers does not find this supports that theory because to him it is apples and oranges, the things we are talking about here are small vessels, and the echocardiogram is looking at heart muscle wall, these vessels are much larger than what we are talking about here. You can have plaque or occlusion in the microvessels without having it in the larger vessels of the heart. That is common. The third thing relied on by plaintiffs is the transcranial Doppler that was taken which states there is free flow in the vessels and all vessels are within normal limits. Dr. Powers states that that does not show Mrs. Hogans did not have microvascular disease in her brain. Why—The transcranial Doppler looks at velocity of flow in the vessel and he has written a bunch of papers on transcranial Doppler, and the weakness of the transcranial Doppler is you cannot pick up small vessels, you are only picking up the main conduits and also the other thing is transcranial Doppler is not an imaging study. It just tells you where the vessel is in the flow, you get no information about the existence of plaque, you get no information about the actual physical structure of the vessel and that is independent of saying you can't see the small perforating vessels, the technology just won't give that to you.

None of the tests have any effect on his opinion that the stroke was caused by hypertension diabetic occlusion of the microvascular vessels of the pons. He agrees with Dr. Swann that the stroke was arterial in nature, no venus outflow obstruction as opined by plaintiff's expert Dr. Rutledge.

You can infer from an MRI a venus stroke as opposed to an arterial stroke based on different distributions.

His opinion within a reasonable degree of medical probability is Mrs. Hogans would have had the stroke that day even if the acoustic neuroma would have been removed or if it had never existed. It was not at all exacerbated by the pressure of the tumor. He explained: Because the part of it is just looking at the anatomy of the blood vessels as I described. The other thing is that - I don't' - the other thing is that there is a pressure phenomenon. And what happens is these tumors expand, or as you put pressured on tissue, the force is not at one point. It is actually distributed. And as you distribute that force, the - actually the tissue has a certain elasticity and can tolerate that. And so as this expands, this force is distributed over a broader area. It seems counterintuitive, but that is what happens in the early phases. So that what happens is this dark - there is no evidence of a pressure phenomenon. I agree that there is a mass there is no doubt about it. There is displacement. But internally, from a pressure point of view, that would postulate. You have to have generated enough pressure to actually affect the vessel. And from a practical

point of view, you know, even with my hands we move the brain, I mean I do that. I don't do it over years, as this thing has been there. I will do it over hours. And so you can put a certain amount of force there, and the tissue can handle it and you don't occlude vessels in that area.

He links Mrs. Hogans' present condition with the surgery that removed the tumor. He stated that in the medical record, there is a one sentence comment that as they were working on the capsule, they lost part of the signal. Now, they put an addendum they said they thought it was a technical problem but it just so happened to be on the side that also affected the arm and the leg. So at the time of surgery they had a baseline evaluation, it was stable through about three quarters of the procedure from what he could guess looking at the timing of it because they didn't document exactly, and near the end of the procedure, they lost it. So something happened at the procedure that had an effect on those pathways and then the whole point is when you see the change, the inference is something has happened to the nerve pathway and that would be consistent with the deficits she woke up with after the procedure. This is a known risk.

He agreed the tumor needed to come out and yes, that is a known risk and that's why you do the monitoring. You know you are going to be working by those pathways that's the whole point of doing it, so if something happens you can try to adjust it to minimize that risk.

What they think happens is that in the act of coagulating inside the capsule, you can have an effect on these vessels and then you can have an effect on vessels that are supplying viable nerve tissue.

He agreed that if the tumor had been imaged and discovered and treated in 1990 then the more serious surgery to the larger tumor would not have had to be done in 2002. But his understanding of the medical record is they were just looking at debulking which means they were looking at just going in and taking the center out, so their original plan was not one of going in there and stripping the capsule down off of all of the adjacent structures, they just wanted to get the mass down.

When asked if it is medically possible for the tumor to have compressed the pons, compressing the blood vessels and leading to strokes, he replied "anything is possible, yes."

In looking at the MRI film of December 21, 2000, he would not be able to show the actual clot in the vessel.

In the tests performed at Harborview they were looking for the source of the clot and narrowing of the vessels but did not find any. Dr. Powers said they looked in the "main vessels." There is also no evidence the stroke was caused by pressure causing an artery

to burst and bleed. He agreed the pons were displaced. The area where the pons meets the brain stem is called the capsule.

He agreed it would be much more preferable to remove an acoustic neuroma from inside the internal auditory canal versus the removal Mrs. Hogans underwent.

The removal of an acoustic neuroma is at a reduced risk than Mrs. Hogans faced in her operation if removed while inside the internal auditory canal.

The risk of injuring the facial nerve permanently is between 3 to 5 percent for small tumors that is why it is better to catch it in its early stages rather than later.

He agreed that when a patient has diabetes or hypertension and it causes a stroke, statistically speaking there are certain areas of the brain where the stroke is going to be caused.

He agreed the least common area for a stroke as a result of diabetes or hypertension would be the area of the pons.

Exhibit 146–the incidence of stroke among people with hypertension and diabetes occur in four areas, one being Basal nuclei, number two being the Cerebellum, and three the Thalamus and number four the pons or brain stem area.

80 to 85% of strokes would be in the basal nuclei and thalamus areas and 10% in the pons and cerebellum.

He agrees the standard of care would have been to do an MRI.

He agreed the reasonable explanation for Mrs. Hogans having a neurological deficit that she has right now is because she suffered the stroke and that placed her at a greater risk for having neurological deficits when the time came to actually remove her tumor that was adjacent to where she had her stroke. He acknowledged he is not an expert in diabetes or in hypertension. Nor is he an expert on a patient with a combination of diabetes and hypertension.

It is his opinion that the tumor did not cause the double vision or the lateral eye gaze. He attributes these symptoms to diabetes. Despite the fact that the medical records show that three days following the removal of the tumor, when the pressure was removed from the pons, the double vision cleared up and the lateral gaze cleared up he does not agree that the more likely explanation for these symptoms was the compression of the tumor on the pons.

His medical opinion that the fact the stroke happened where it did is just coincidence and that if the tumor never existed, Mrs. Hogans would have had the same occurrence on the same day.

The difference between the information the doctors had initially and what Dr. Powers had is the MRI and her recovery. If this was a pure pressure phenomenon and it was occluding vessels because of pressure, those vessels are not going to revascularize and her neurologic status would have remained stable if not deteriorated. So the fact that there was improvement in neurologic function tells him there was still blood supply to that area and that's the pattern that one sees with a stroke. Had it been a pressure phenomenon, if you had enough to occlude a vessel which is questionable, then the – it would still be there and there would never be any recovery of any of her neurologic function.

If the plaintiffs are correct and the pressure from the tumor caused the stroke in the pons, Mrs. Hogans would not have recovered because the tumor wasn't removed. The occlusion still would have been there.

He has removed bigger acoustic neuromas and has never seen a stroke associated with them. The tumor in this case was debulked from his reading of the record-- they went in and they just worked in the capsule and took out the central portion. This is a safer surgery than resection.

He has never seen an article that attributed a stroke like Mrs. Hogans suffered to an acoustic neuroma.

The wedge pattern of the infarct is not consistent with pressure from the pons. It is consistent with occlusion of a paramedium branch off the basilar artery and that what you get when you have hypertension and diabetes.

3. Dr. David Musselman–Board Certified Cardiologist.

Cardiovascular disease is the development of plaque or atherosclerosis generally in the arterial system that supplies various organ systems around the body. Cardiovascular disease comprises the entire vascular system. There is a large overlap between the risk factors for cardiovascular disease and risk factors for stroke.

In reviewing Mrs. Hogans medical records, his opinion is Mrs. Hogans had significant risk factors for cardiovascular disease. Her risk factors were diabetes, severe uncontrolled hypertension, obesity, physical inactivity and high cholesterol. He believes that once someone has had a stroke, their risk of subsequent stroke is significantly increased. Based on the medical records, Mrs. Hogans may have had some of the risk factors as far back as 1982. The length of time a person has a risk factor increases the risk. The combination of risk factors increases the overall risk of stroke.

In reviewing Mrs. Hogans' medical records, he believes her compliance was poor. He acknowledges in his deposition he opined her compliance was average but in re-reviewing the medical records, it struck him that the references to noncompliance were really more pervasive than he had recalled at the time of the deposition, and he was particularly struck by one particular entry where there was, he believes, in January of 1989, where there was a medical record where a three-year period of noncompliance with antihypertensive medications was reported. Compliance with recommended therapy is designed to reduce risk. Reducing weight, reduction of blood pressure, improvement of someone's cholesterol is all designed to reduce risk, and if those treatment recommendations are not followed, one's risk increases.

As a practicing cardiologist he rated her risk for stroke in December of 2000 as very high, "her stroke would almost have been, potentially foreseeable." In his deposition he was asked to quantify the percentage of risk for a patient with hypertension and diabetes. He guessed it was between 3 to 5 percent, and he was referring to absolute risk. Absolute risk would be an individual's chances of having some type of event over a defined period of time.

Hypertensive strokes are usually ischemic. An echocardiogram is an imaging study of the heart that uses a sonagraphic technology.

Government's Exhibit 2 is a echocardiogram performed December 26, 2000, at Harborview Medical Center. The impression indicates there is left ventricular hypertrophy-- a thickening of the heart muscle. There are numerous causes of left ventricular hypertrophy, the most common is hypertension. As a response to hypertension.

End organ damage is when there is one pathological state that damages or makes a physiological change in a secondary organ. Left ventricular hypertrophy is a type of end organ damage and is associated with hypertension. The echocardigram also reveals relaxation abnormality-described as follows: there are two phases to the cardiac cycle, systole is when the heart muscle contracts and dystole is when the heart muscle relaxes and if the lining of the heart is impaired, if the heat muscle is stiff, and requires a higher pressure to fill in dystole, that is what a relaxation abnormality is, and is frequently seen in left ventricular hypertrophy and it is a result of hypertension.

Given Mrs. Hogans' risk factors, he would expect her to have a reduced life expectancy.

He agreed he does not have particular expertise in the cause of strokes. He agreed that in spite of a patients' best efforts, there are some that hypertension still remains elevated. He thinks Mrs. Hogans had very difficult to treat and very difficult to control hypertension and she was on multiple anti-hypertensive medications and in spite of being on those multiple medications, she still had significant hypertension.

He thinks it unlikely that had she been fully compliant with all the lifestyle changes it would have taken care of her hypertension.

He is not rendering any opinions about the cause of Mrs. Hogans' stroke. He was asked to comment on her cardiovascular risk factors and the potential contribution that had to her stroke.

4. Dr. Julio Chalela----Board Certified in Neurology–his specialty is as a stroke neurologist.

Stroke neurologists are involved in the prevention, diagnosis and treatment of ischemic and hemmorrhagic strokes.

He described the difference between a general neurologist and a stroke neurologist as follows: A general neurologist has completed a residency in neurology which is technically four years in most programs, a stroke neurologist in addition to the general neurology residency, has done a fellowship anywhere from one to three years of training limited exclusively to the field of stroke.

He did his fellowship: 2 years at the University of Pennsylvania and an additional year at Johns Hopkins University Hospital.

He has training in reading MRIs and CTs.

In his practice, he almost exclusively sees patients with strokes.

When called in as a stroke neurologist, he obtains a history from the patient, he performs a physical and a neurological exam, he orders tests that are considered pertinent either the CT or MRI and ultrasound–and then with a clinical history, the risk factors and the findings and the imaging studies, he makes his diagnosis and comes up with a plan of treatment.

He does not agree with plaintiffs' theory in the case that Mrs. Hogans' acoustic neuroma grew to such a size that it put pressure on the pons occluding veins or arteries and causing a stroke.

He states the plaintiffs' model showing the mass effect with the golf ball is unscientific.

He believes Mrs. Hogans' stroke was caused by small vessel ischemic disease related to hypertension and diabetes.

He defines small vessel disease: Small vessel disease, small ischemic disease is the progressive blockage and narrowing of the blood vessels that feed the deeper, inner parts of the brain, related mainly to hypertension and diabetes, smoking, high cholesterol. As

opposed to large vessel disease which is blockage of the big blood vessels like the ones in the neck, small vessel disease refers to tiny, small blood vessels that we can't see with any of our imaging studies in the deeper parts of the brain.

A person can have small vessel ischemic disease without having ischemic disease in the large vessels. This is common.

He was asked about Dr. Swann's testimony where Dr. Swann testified that he had ruled out small vessel disease due to hypertension and diabetes based on three tests. Dr. Chalela states the MRI provided some evidence Mrs. Hogans did have small vessel disease in other parts of her body. He explained: Some small lacunes, remote from the brain stem stroke, and also there is a neurologic examination performed at the hospital in Seattle that indicated that she had loss of pinprick sensation on her feet, which is suggestive of a peripheral neuropathy, likely related to diabetes, secondary to small vessel injury to the nerves that go down her legs.

Dr. Chalela found this related because hypertension and diabetes affect more than one organ. For instance, in Mrs. Hogans' case it affected her heart and she has left ventricle hypertrophy, diabetes seems to have affected her feet, and than a combination of diabetes and hypertension has affected her – the blood vessels in the brain. So it is a multisystemic

The transcranial Doppler is an ultrasound examination but does not look at the smaller vessels.

It is not uncommon to have free flow in the large vessels and still have occlusion in the small vessels. The carotid Doppler only rules out larger vessel disease.

The three tests performed did not rule out hypertension and diabetes as a cause of small vessel ischemic disease in Mrs. Hogans. It is his opinion that Mrs. Hogans had a stroke in the pons due to ischemic vessel, small vessel ischemic disease. He bases his opinion on the clinical presentation of, on the risk profile, and on the MRI findings.

The clinical picture: the fact that the symptoms presented sort of in a stuttering fashion over a couple of days before she had the stroke, she had some transient left handed morsesis and left hand weakness that got better and came back the next day.

The sort of slow progression is suggestive of small vessel disease. The rapid or rather the rapid recovery that followed the stroke where she got significantly better after she had the stroke is also a clinical feature that is common with small vessel disease.

As to the stuttering, Dr. Chalela explained: It has been recognized for decades by stroke neurologists and sort of the classical clinical presentation or the characteristic clinical presentation for small vessel disease and it is believed that either there is a clot or there is

an "athroma, a piece of fat buildup that is blocking the entrance of a blood flow into a blood vessel. And the symptoms are coming and going as the obstruction is occurring intermittently.

Because of the slow progression instead of a catastrophic onset with progression over seconds, this slow progression matter of minutes to hours has been described as a characteristic clinical presentation for small vessel ischemic disease.

Another factor that makes Dr. Chalela think it was a small vessel ischemic stroke rather than a compressive stroke was her rapid recovery. From what he read in the record, in the months following the stroke, she got significantly better and was able to ambulate. This is described as a characteristic feature of a small vessel ischemic stroke that patients tend to get better as opposed to, for instance when you have a complete blockage of your carotid artery you suffer a stroke that involves the whole right side of the brain for instance, you don't' get better in the next few months.

That indicates it was not caused by pressure from the tumor because the tumor was still there. He would not expect the pressure to be relieved just spontaneously. The tumor was removed later on, and the patient got worse after the removal of her tumor. The pressure from the tumor was not released after her stroke.

The MRI also supports his theory. It shows an area of increased signal intensity which is the white area. The right side of the pons. The tumor is on the right side. He was reminded that Dr. Swann testified that the area of increased signal intensity was the stroke. Dr. Chalela responded: Well, hindsight is always 20/20. But looking at this MRI, what one would think that but looking at the follow-up MRI that was performed later on which I will show you in a little while, it becomes obvious that part of this white this was swelling that this appeared and then actually the stroke was just a small area right here towards the center of the brain stem. And that most of this area actually disappears over time. If it were truly, if all of this were stroke, what I would have expected that when follow-up studies were performed that area would still be abnormal. And, in fact, the new radiologist that read the MRI in Seattle clearly stated that most of the lesions seen on MRI were gone when follow-up MRI was performed.

The entire area seen on the MRI was not actually stroke because it went away.

He explained: So if the stroke were caused by the tumor, which is an immediate proximity to the lateral aspect of the brain stem, I would expect this abnormal signal to be right here, but it is actually remote from the tumor. It is more towards the middle part of the brain stem.

When asked why would you expect the stroke to be adjacent to the tumor, he responded: Because if we are trying to invoke the tumor as the mechanism that causes stroke, the

perforating arteries that encase the brain stem sort of go right around it like this and wraparound the brain stem, and if the tumor were going to squeeze those blood vessels, it would do it right there and would compromise the blood flow to the lateral aspect of the brain stem right there. Because that is where the blood vessels would be squeezed. There is arterial blood flow around the outer portion of the pons.

What he is saying is that the pons has arteries on the outside of it that if you were pressing on, those would constrict the arteries on the inside of the pons.

He also thinks the most important aspect of what was being discussed about th image: that the neuroradiolgist who read the studies also commented on, if you look at the follow-up MRI which was done on March 8, 2001, thee tumor is still there. But most of that abnormal signal seen before is gone and in fact, you just see some very faint white patches in the more central part of the brain stem, not in the lateral part where the tumor is. It is actually very hard to see if you are not used to reading MRIs but you see some of it right here but most of the pons now have normal appearance with the exception of the tumor being right there.

If it had been just pressure on the pons from this tumor and all of that was seen before, the abnormal signal was actually stroke, it still should be there. Now, most of it is gone, most of what is in the – what was machine before on the lateral aspect of the brain stem is now gone and what we are left with is some very subtle signal in the central part of the brain stem.

Another factor on which he bases his conclusion is the fact there are multiple small strokes elsewhere in the brain.

Mrs. Hogans definitely has microvascular disease throughout her brain.

Also the result of the evoked responses during surgery suggests that the mass effect from the tumor was not that significant because the evoked responses were actually normal until the surgery was completed which indicates that the electrical stimulus was going all the way up through the nerves, through the spinal cord, through the brain stem and making its way up to the brain. If there had been significant mass distortion and mass effect on the brain stem, the evoked response would have stopped at the brain stem. Dr. Chalela states it is not a major mass effect because it has not interrupted the electrical activity of the brain stem.

Venus infarcts are usually hemorrhagic. The vein is interrupted or blocked, pressure builds up within the vein, the vein has a fragile wall, and it bursts and it causes a hemorrhage. They usually have a slow gradual course that happens over a matter of weeks, they are usually associated with headaches and they usually don't happen in the brain stem, they usually happen in the top part of the brain.

He performed a medical literature database search engine. He tried to link stroke or cerebral vascular disease with acoustic neuroma . He searched from 1964 to the present day, 2004 and did not find any. He did the search in Spanish and English.

It is his opinion within a reasonable degree of medical certainty that Mrs. Hogans suffered a stroke due to small vessel ischemic disease as a result of her longstanding hypertension and diabetes.

He would interpret what happened during the surgery is a result of the neurosurgeons having to retract or pull aside the nerves, the temporal lobe, so it is not unusual to have brain injury as a result of this surgery.

On cross-examination he was asked if it was just a coincidence that the tumor is right adjacent to the brain stem in the pons and it just so happens that of all the places in the body that Mrs. Hogans can have a stroke she happened to have a stroke right there? He responded: Actually the stroke is not right next to the tumor, the stroke is medial in the medial part of the brain stem, the tumor is lateral to the - to where the tumor is,. And it is just not — not a coincidence. The - strokes are actually quite common in the pons in this part of the brain because of the anatomy of the blood vessels in this part of the brain actually they have been called the arteries of stroke so it is not a coincidence and the tumor is not next to the stroke. The stroke is medial.

He agreed an acoustic neuroma can cause a patient to have intermittent symptoms related to the nervous structures, that the tumor is compressing but not intermittent vascular symptoms which is very different and which is what happened preceding the onset of her stroke and a couple of days before the onset of the stroke, so that is a very different scenario when you are describing pressure or compromises of the nervous structures than when you are compromising the vascular structures.

Although he agreed that it was possible that the tumor put pressure on the blood vessels and occluded the vessels–everything is possible in life, but that is not what accounted for Mrs. Hogans' stroke because if that had been the case, the blood vessels that would have been compromised would have the lateral, short circumferential branches which are on the side of the brain stem. In this case, the blood vessel that was affected was a blood vessel that was coming "front and medial part of the brain stem.

The four tests were normal with respect to any narrowing or blockage with respect to the large vessels. The technology does not exist to see the small vessels.

On cross, Dr. Chalela was asked to agree that at least 4 attending physicians' statements written directly into the record attribute the stroke as a result of the tumor compressing the pons and the vessels in the pons. In response, Dr. Chalela remarks there is also a note from Dr. Winn who is a neurosurgeon who saw her in Seattle--he states that the brain stem

dysfunction that the patient has is actually secondary to vascular disease. So there are attending physicians that saw her that agreed with Dr. Chalela's opinion. These doctors in the days after her stroke attributed the stroke to the tumor putting pressure on the pons. Dr. Chalela states that those doctors did not have benefit of seeing the followup MRI that was performed 3 months later which would change the analysis.

About 25% of small vessel strokes happen in the pons.

Dr. Winn's letter, which is Government's Exhibit 2, states the purpose of the surgery is principally to get pressure off the brain stem where she had significant dysfunction either related to the tumor itself or to vascular disease--which is the point that even Dr. Winn agreed there could be vascular disease causing her symptoms.

None of the attending physicians were subsequently deposed to give them an opportunity to "correct their earlier statements" once and for all.

In making the diagnosis of small vessel ischemic disease, you have to rely on all other possibilities so you arrive at that conclusion after ruling out cardiac disorders, blockage in the neck, blockage of the basilar artery and seeing that clinical pattern and the MRI pattern, then you come to a conclusion that it is small vessel disease

Doctors use the term secondary to mean trying to determine causation.

He excludes the tumor as causing the stroke as there is clear evidence from the clinical symptoms, the MRI findings from the progression of the symptoms and from her recovery that this was secondary to the hypertension and diabetes. The surgery was not a contributing factor to the stroke.

Her condition was exacerbated by the subsequent surgery. She already had a brain that was injured because of a small stroke. And on top of that you had the injury from the surgery that sort of magnifies her injury.

He believes it was mere coincidence in terms of the proximity of the tumor and the stroke. If you look at the follow-up MRIs, the tumor and the stroke are not in close proximity. The tumor is on the right side of the brain and the stroke is in the central part or towards the center of the brain stem. It is not in immediate contact with the tumor.

5. Dr. Kevin M. McGrail - board certified neurosurgeon. He is the Chief of Neurosurgery at Georgetown University Medical Center and School in Washington D.C. He is Chairman of Georgetown's Department of Neurosurgery.

Dr. Swann trained at Massachusetts General. He also knows Dr. Yu when he was a medical student at Harvard. Dr. Yu is very qualified and he respects Dr. Yu and knows him professionally.

He teaches residents and interns and sees patients and practices neurosurgery in the operating room. His focus is on cerebrovascular disease and brain aneurysm. He currently has patients with acoustic neuromas and over his medical career, including training, he has probably had several hundred patients with acoustic neuromas.

An acoustic neuroma is a fairly common tumor; it is the most common benign tumor in adults in the posterior cranial fossa. The typical signs of an acoustic neuroma: unilateral hearing loss, tenitus or ringing in the ear, unsteadiness or ataxia.

As a neurosurgeon, he would have put acoustic neuroma on his differential diagnosis for Mrs. Hogans.

A normal CT scan with contrast does not rule out that Mrs. Hogans might have had an acoustic neuroma. The magnetic strength of MRIs varied drastically from center to center during the time frame in issue.

Many people with acoustic neuromas larger than Mrs. Hogans have never suffered a stroke caused by the acoustic neuroma. He cannot find Mrs. Hogans' stroke was caused by the acoustic neuroma where there is not one reported case of such an event, while on the other hand, it is common for people with Mrs. Hogans' risk factors to suffer strokes.

He cannot make the statement that if an MRI had been done in Mrs. Hogans' case that more likely than not she had an acoustic neuroma at that time. He can say that more probably than not she had the acoustic neuroma in the early 1990s.

He has seen tumors much larger than Mrs. Hogans' put much more force on the brain stem and arteries/perforating vessels and there is not a report case where it caused a stroke. The much more likely cause of Mrs. Hogans' stoke is her well known and accepted risk factors for stroke.

He agrees the standard of care in July of 1992, was for an MRI to have been done.

He believes Mrs. Hogans suffered an occlusion of an artery probably of a significant branch off of the basilar artery and that the occlusion was caused by atherosclerotic disease. Mrs. Hogans' acoustic neuroma had nothing to do with her stroke.

He cannot say that plaintiffs' theory the acoustic neuroma put pressure on the blood vessels cutting off the blood supply can never happen but is certainly is not a common event. This patient had two serious risk factors for brain stem stroke. More likely than not the acoustic neuroma was not the cause of the stroke. However, he can never say it is absolutely impossible.

The existence of the two risk factors would prevent his case being published as a case report.

Had Mrs. Hogans' acoustic neuroma been diagnosed in February or March of 1990, she would have had three treatment options: serial MRIs to observe the tumor over time, surgical removal of the tumor, or stereotactic radiosurgery. The option chosen is usually based on the patient's choice.

He had no criticism of the doctors wanting to do the debulking surgery.

He prefers to operate on a smaller tumor because the surgery is technically easier to do, the surgery is of a lower risk. Most tumors of Mrs. Hogans' size are typically resected. This case is unusual. With smaller tumors, the surgery does not take as long, the surgeon can see the nerves better and there is less danger because you can see the blood vessels better.

He stated that if the tumor had been removed in the 1990 to 1992 time frame he could state Mrs. Hogans' would not have been affected as she currently is – this is a risk of surgery. He claims he has seen every neurological catastrophe that can occur from surgery.

He would not have predicted what happened to Mrs. Hogans to have happened.

He agreed a patient with stroke risk factors can live his or her entire life without a stroke–it just means he or she is at an increased risk. Mrs. Hogans has two major risk factors so she is at an increased risk for stroke.

He believes the proximity of the tumor is a coincidental occurrence. The location of Mrs. Hogans' stroke is not an uncommon place for a stroke. The medical literature is replete with support for his opinion that someone with Mrs. Hogans' risk factors is likely to suffer a stroke.

He agreed that once information is placed in a medical charge, that information can be perpetuated.

When asked if he was making a "jump" by saying that hypertension and diabetes was the cause of Mrs. Hogans' stroke, Dr. McGrail replied: "I don't think that is any jump at all. I think that's well supported in the medical literature. I think the jump is to say that this was caused by the tumor. That's an enormous jump. And so I think that – I think my opinion is – is well founded and supported by what is known about stroke, diabetes, hypertension. The big jump is to say that this was secondary to the tumor. There is – I couldn't find a single case report of that in the literature. So you know, I suppose anything is possible, but there is just – that's – that, to me, is the opinion that's the most speculative."

APPENDIX "B"

1. Dr. Lee M. Ratner - the government employee radiologist who did the CT scan in February of 1990.

Attended Albany Medical College in Albany New York; residency in diagnostic radiology. Was at Malcom Grow at Andrews Air Force Base in Washington, D.C.

During the time from from 1985 to 1989, Dr. Ratner agreed that he was using MRIs as part of the diagnostic tool to diagnose acoustic neuromas. He in fact saw an acoustic neuroma on an MRI during his residency.

He agreed that as the tumor grows some of the possible effects are it can compress nerves and arteries. It can put pressure on structures surrounding the tumor. Dr. Ratner thought if one of the surrounding structures is an artery as the tumor grows it can put pressure on that artery.

He agreed with counsel's translation of a sentence that meant that the tumor mass is having an effect on the arteries such that it is occluding the arteries and causing an infarct in the brain.

Referring to Exhibit 31, Dr. Ratner agreed that what the radiologist was saying was as the acoustic neuroma was growing it is putting pressure on those arteries that are going inside the substance of the brain. He stated it was an assumption that it is causing an infarct on the brain.

He stated that if an acoustic neuroma is inside the internal auditory canal it is very difficult to see with a CT. He acknowledged that a patient can actually have an acoustic neuroma located inside the internal auditory canal and the CT, even with contrast, would be read as normal. He also agreed that if a physician does a CT on a patient with a suspected acoustic neuroma and the CT comes out normal, it does not mean that an acoustic neuroma has been ruled out. An enhanced MRI is a more sensitive way of picking up an acoustic neuroma and was the most sensitive test in 1990 of any test at picking up an acoustic neuroma.

The CT on Mrs. Hogans was performed on February 22, 1990, at Andrews Air Force Base, Malcom Grow Medical Center. Dr. Ratner was unable to see an acoustic neuroma by this CT. Dr. Ratner recommended that a MRI be done in 1990 on Mrs. Hogans.

2. Dr. Diane Sommer – government employee radiologist involved in Mrs. Hogans' treatment —called by plaintiff as an adverse witness.

After the July 24, 1992 visit, Dr. Sommer ordered a CT scan because of light headedness and uncoordination and numbness. The hearing loss on the right side was an added factor but what made it urgent was the light headedness and the unsteadiness.

Dr. Sommer looking for a mass or lesion that somehow was affecting the 7th cranial nerve among other things, and the CT scan came out negative. She other ordered other tests including a complete blood count and a profile A and B. After these tests, she did not have a definitive diagnosis.

If the acoustic neuroma was inside the internal auditory canal and was putting pressure on the 8th cranial nerve and also the facial nerve, the 7th cranial nerve, that could account for Mrs. Hogan's symptoms as of the 24th and 30th of July, 1992.

She agreed if an enhanced MRI study had been done of Mrs. Hogans from any point from February 22, 1990 through February 22, 1993, it could have revealed Mrs. Hogans had an acoustic neuroma. The most likely explanation for the hearing loss and the facial numbness is an acoustic neuroma.

Dr. Sommer first treated Mrs. Hogans on June 6, 1991, for blood pressure medicine refill. Therefore, Dr. Sommer would not normally look at three years of her chart.

Risks for stroke are: hypertension, diabetes, smoking, obesity, elevated cholesterol, inactivity and prior strokes. Race is also a factor–African Americans have a higher risk. Concerned about diabetes because it causes microvascular disease. When Mrs. Hogans came in to see Dr. Sommers on June 6, 1991, her blood pressure was 191 over 103. The normal range is 140 for the top number and the bottom number back then was 90. Mrs. Hogans' weight was 204. On a return visit on December 5, 1991, her blood pressure was 194/86 and her weight was 206. On January 22, 1992, her blood pressure was 152 over 94 and her weight was 198. February 11, 1992, her blood pressure was 174 over 88 and her weight 198½. Later in the visit, her blood pressure was 160 over 90. Her next visit was February 26, 1992. Her blood pressure was 184 over 85 and when retaken later was 150 over 85. On May 5, 1992, her blood pressure was 198 over 88 and her weight was 202.

When Dr. Sommer ordered the CT scan she was looking for a mass or lesion that somehow was affecting the 7th cranial nerve and she was also looking for stroke. Her symptoms made her think she may have had a stroke. A CT is better at detecting a stroke than an MRI.

3. Dr. Charles Ford - by video - government employee resident physician in otolaryngology who treated Grace Hogans in 1990.

Dr. John Kopp was his supervising physician at the time he was doing his residency.

Dr. Ford agreed the reason he ordered a CT of the posterior fossa with contrast on Mrs. Hogans was to rule out an acoustic neuroma. He also knew that in January of 1990, a CT would not pick up an acoustic neuroma contained within the internal auditory canal. In principle, he agreed that the prognosis is good if an acoustic neuroma is caught early and treated.

4. Dr. John D. Kopp - government employee physician and Chief of Otolaryngology at Malcolm Grow Medical Center/

It was a matter of filing out one or two forms to get an MRI done at Malcolm Grow. Dr. Kopp agrees that even if a CT test with contrast is run and is read as normal, it doesn't necessarily rule out an acoustic neuroma. An acoustic neuroma can be inside the internal auditory canal. If the acoustic neuroma is growing outside the internal auditory canal towards the brainstem, as its growing outside the canal, it will have a mass effect on the surrounding structure. And a mass effect means as it's growing towards the brainstem, it will start compressing the structures surrounding the tumor. It can compress both nerves and arteries. It can also compress veins and ventricles of the brain.

The report they were looking at said that the mass that's located at the CP angle on the right hand side is occluding perforating arteries of the brain.

When asked what the radiologist in the report, Exhibit 1, says is the most likely cause of the dead tissue in Mrs. Hogans' brain, Dr. Kopp responds it is most likly secondary to occlusion of the perforating vessels by the right CP Angle mass. When asked what does that mean, Dr. Kopp responds that the blood vessels in the area are occluded by the mass. Dr. Kopp agreed the radiologist is not saying that the infarct is caused by hypertension, diabetes, or any other causes other than the occlusion of the vessels.

Dr. Kopp agreed that in 1990 and today, it is true that a gadolinium enhanced MRI was more sensitive at picking up an acoustic neuroma. He also agrees that the radiologist is making a recommendation to the physician that, if they are looking for an acoustic neuroma, to do a gadolinium enhanced MRI. He agrees that in February of 1990 when the CT scan was read as normal and the radiologist recommended that an MRI be done, if the doctor was looking for an acoustic neuroma, an MRI should have been done.

He acknowledges he was the head of the department of otolaryngology at Malcom Grow every time Mrs. Hogans was seen there.

He also acknowledges that no doctor ever noted, in the medical records that he reviewed, Mrs. Hogans' symptoms of hearing loss, ringing in her ears, ataxia, dizziness with imbalance, twitching of her right eye and the right side of her lip, numbness on the right side of her face that comes and goes, was attributed to diabetes or hypertension.

Mrs. Hogans' hearing loss had increased between 1990 and 1993. An MRI should have been ordered. He stated the hearing getting worse may have been a sign of acoustic neuroma being there and getting larger. Acoustic neuromas are very slow growing. He agreed the earlier you diagnose a patient with an acoustic neuroma they better the outcome. He agrees that after the audiogram was done in 1998, an MRI definitely should have been done at that point because her hearing had gotten worse. He agreed if an MRI had been done in 1993, it is more likely than not the acoustic neuroma would have been picked up. He also agreed the prognosis is good if an acoustic neuroma is caught early and treated appropriately.

APPENDIX "C"

1. Dr. Laurence Cambron, attending Neuroradiologist at Harborview Medical Center, stated the following upon his review of the MRI: "Right CP angle mass is having marked mass effect on the pons. Foci of hyperintense diffusion signal consistent with acute infarct, most likely secondary to occlusion of perforating vessels by the right CP angle mass."

2. An attending Neurologist at Harborview Medical Center stated upon review of the MRI and examination of Mrs. Hogans: "The most likely etiology of the infarct is compressive occlusion of the penetrating vessels."

3. Dr. Stephen Settle, board certified in Physical Medicine and Rehabilitation made the following statement: "Assessment: Cerebrovascular accident secondary to right cerebellar pontine tumor infarct."

4. Dr. Chan Hwang, also board certified in Physical Medicine and Rehabilitation stated: "Impression: 1. History of right pontine stroke with left hemi paresis. 2. Actually the stroke was secondary to a right cerebropontine angle schwannoma which was resected on May 3, 2002."

5. Dr. H. Richard Winn - Neurosurgeon-in-Chief at Harborview Medical Center

   In his letter to the referring doctor, Dr. Turella from the Madigan Army Medical Center, dated December 27, 2000, Dr. Winn wrote: "We concurred with your diagnosis of brainstem stroke which is close to where her tumor is located. She is making significant improvement and when she stabilizes, we would consider either surgical approach or treatment with our GAMMA Knife. In addition, we had her seen by our stroke neurologist Kyra Becker, M.D."

   Dr. Winn wrote to Dr. Turella again on March 14, 2002, after seeing Mrs. Hogans the previous day. He wrote:

   > As you know, she is a 55-year-old woman with a known right CP angle tumor, most likely a vestibular schwannoma. I initially saw her more than a year ago, when she presented with symptoms suggestive of a brainstem stroke. She originally was admitted to Harborview Medical Center on the 21st of December and had right facial findings and a left hemiplegia, which improved with time. There were changes in her brainstem suggestive of edema and/or ischemia in proximity to the mass. I chose not to proceed with a surgical decompression, because I thought that the risks were quite

high that the ischemic event would either be made worse, or her recovery from her stroke would be impaired. Over the past year, she has gone from being wheelchair bound to walking without a cane and has recovered function more so in the lower extremity than the upper extremity. Between March and June of last year, she would awaken with some diplopia, which would clear within a matter of a few minutes to a few hours.

However, beginning on March 10, 2002, when she awoke at 4 a.m., she had diplopia, which persisted. In addition, she and William felt that her ability to walk was worse. This morning, she did not awaken with diplopia, but noted its onset around 10:00 a.m.

As you are aware, she was seen and evaluated by Drs. Enquest and yourself, and subsequently by Dr. Scott. The latter noted a subtle left 6th nerve paresis without any other cranial nerve or long track signs.

To my examination, there was diplopia with left lateral gaze, and I concur with Dr. Scott's findings. The rest of her examination reveals residual left sided spastic hemiplegia, more so in the distal aspects of the left extremities and more so in the upper compared to the lower extremity. She has developed a cataract in her left eye and presently has 20/40 vision, where as in the right it is 20/20.

In my office I had a long and detailed discussion with the Hogans and indicated to them that I did not think these symptoms related to her mass, but rather more probably relate to a small stroke, perhaps secondary to her diabetes. Primarily I would base this on the fact that she has a very mild left 6th nerve palsy, whereas the mass is on the right. Surprisingly, she has no other new brain stem findings, apart from a history of change in her gait.

Our original plan formulated last year, was to await her recovery and then consider either excision and/or GAMMA Knife treatment for her mass. I think we should allow her at this point to recover from what presumably is a small stroke. If on the other hand, her symptoms progressed, then it might well be related to the mass, despite my reservations noted above.

However, I am hopeful that she will recover and in 4-6 weeks. We should then contemplate a surgical approach in which we de-bulk the tumor, but with a strategy that we would not attempt a total

> resection and place her 7th nerve in jeopardy. This internal debulking procedure would avoid a prolonged operation that might compromise her tenuous vascular status. Following this internal decompression, we would plan, after several months, to treat the residual tumor with our Gamma Knife. The surgical internal decompression would allow the tumor to move away from the brainstem, so that the Gamma Knife Radiation could be more safely applied.

The following paragraph was contained in a letter to Dr. Turella dated April 26, 2002:

> At this pint, I think it is reasonable to consider going forward with the debulking of her tumor, which presumably is a 5th nerve neuroma. The purpose is principally to get pressure off of the brainstem, where she is had [sic] significant dysfunction either related to the tumor itself or to vascular disease. She and her husband fully recognize the options, risks, and benefits of such an approach and I reviewed with them again, as I have done many times in the past, the risks of the surgery. They fully understand and desire to proceed.

In a second letter also dated April 26, 2002, Dr. Winn made the following observations to Dr. Turella:

> In regard to radiosurgery of the residual intercanalicular tumor, I would recommend deferring consideration of radiosurgery or radiotherapy until there has been more time so that she could regain more neural function. Radiation could potentially impair recovery of the 7th nerve. Moreover, assuming at some point she would undergo radiation therapy, staged radiotherapy might have a lesser chance of damaging her right 7th nerve, which was impaired by surgery. Moreover, in the setting of a longstanding diabetic whose cranial and peripheral nerves had fragility related to micro vascular disease, radiation could further damage her seventh nerve.

6. Dr. Jeffrey G. Jarvik, a radiologist at Harborview Medical Center provided the following report concerning the MRI performed on Mrs. Hogans on March 8, 2001:

> No change in right CP angle tumor, likely representing vestibular schwannoma. .
>
> Near complete resolution of signal abnormality in right pons secondary to ischemia/infarction.

. . . .

An intensely enhancing extra-axial cerebellopontine angle mass is again seen. This measures approximately 1.6 x 2.7 x 2.2cm and extends from the cerebellopontine angle into the internal auditory canal on the right. The size, configuration and enhancement pattern of the mass remains unchanged since the comparison examination. This likely represents a stable vestibular schwannoma.

There continues to be mass effect seen on the pons and medulla, which is unchanged. However, the abnormal high signal seen on the diffusion weighted images previously in the pons have now resolved. The abnormal signal that was seen on the FLAIR has markedly decreased, and now there is only punctate area of high signal, likely representing residual area of infarction/ischemia.

Scattered areas of high signal on FLAIR is seen in subcortical and periventricular white matter, which are unchanged and likely represent sequela of small vessel ischemic disease. The remainder of the brain appears normal.

APPENDIX "D"

Plaintiffs' Experts:

1. Dr. Don Huddle—a Professor Emeritus of Economics at Rice University, for head of the Department of Economics at Rice University, and has been teaching economics for over thirty years.

   The appropriate discount rate for reducing Mrs. Hogans' future economic losses in the form of medical costs of 1.2%. He arrived at this discount rate by averaging short, medium, and long term United States Government Bond rates (6 months to 24 year U.S. Government Bonds) resulting in an average of 3.7%. He subtracted from this percentage the current inflation rate (CPI) of 2.5% to arrive at a discount rate of 1/2% for future medical costs.

   He utilized short, medium, and long term government bond rates extending up to 24 years because Mrs. Hogans' statistical life expectancy is 24 remaining years and her future medical care will be delivered over her life expectancy.

   The appropriate discount rate for reducing Mrs. Hogans' future economic losses in the form of lost earnings and earning capacity is 1.0%. He arrived at this discount rate by averaging short, medium and long term United States Government Bond rates (6 months to 8 year Government Bonds) resulting in an average of 3.5%. He subtracted from this percentage the current inflation rate (CPI) of 2.5% to arrive at a discount rate of 1.0% for future lost earnings and earning capacity.

   He utilized short, medium, and long term government bond rates extending up to 8 years because Mrs. Hogans' work life expectancy is 8 remaining years.

   His method of averaging short, medium, and long term government bond rates then deducting the rate of inflation follows the "below market" discount rate method required by the Fifth Circuit as stated in Culver v. Slater Boat Co., 722 F.2d 114 (5th Cir. 1983). His discount rates of 1.2% for future medical costs and 1.0% for future lost earnings and earning capacity fall squarely within the example of proper below-market discount rates set forth in Culver.

   The discount rate method has been accepted in similar severe brain injury cases tried to the Court in the Western District of Texas. Specifically, the discount rate method was accepted in Dickerson v. United States, a Federal Tort Claims Act medical malpractice case tried before United States District Judge H.F. Garcia of the San Antonio Division of the Western District of Texas in 1999. The discount method was accepted by Austin Division United States District Judge James Nowlin in 2000 in a Federal Tort Claims Act medical malpractice case styled Lebron v Untied States.

The methodology employed by defendant's economic expert, Dr. Stan Smith, violates Culver because Dr. Smith relied primarily on a market methodology specifically disapproved by the Fifth Circuit Court of Appeal's below market requirements.

Dr. Smith's high discount rate of 7.42% fails the Culver test because Dr. Smith's methodology employs a 2/3 (65%) reliance on the stock market's average over the highest performing period in the market's history, and Culver mandates a below market method that distinguishes a seriously injured plaintiff's need to sustain his or her future economic needs after suffering a serious injury from speculating investors willing and able to accept some risk for a potentially higher return on their invest.

The present value of Mrs. Hogans' life care plan prepared by Dr. Willingham is $3,327,432.00.

Mrs. Hogans' past lost earning capacity and lost services is $161,842.00.

The present value of Mrs. Hogans' future lost earning capacity and lost services is $611,505.00.

2. Dr. Erin Bigler - board certified neuropsychologist.

Mrs. Hogans suffered permanent cognitive as well as physical injury, and will not be capable of working against as a result of her brain stem injury.

Mrs. Hogans has endured and will continue to endure into the future, physical pain, mental anguish, physical impairment, mental impairment and disfigurement as a result of her brain stem injury.

Mrs. Hogans' condition is as represented in plaintiffs' exhibit 95. Both the left and right side of her body are impaired. Mrs. Hogans will not likely ever walk in a functional way.

There was no cognitive decline in Mrs. Hogans before the stroke.

Mrs. Hogans' cognitive impairment resulted from her brain stem injury. As a result of this injury, Mrs. Hogans' cognitive function decreased from the superior range to the fifth percentile in some areas. In other areas, her cognitive function was at or near the average range, a much lover level than her pre-stroke cognitive functioning level.

Mrs. Hogans suffers from short term memory loss, problem solve decline, and impaired complex cognitive functioning as a result of her brain stem injury.

Mrs. Hogans experiences physical pain and discomfort as a result of her brain stem injury. She is in a wheelchair for long periods of time and as a result, experiences a certain degree of physical discomfort.

Mrs. Hogans has a full range of emotions and understands she has a lifelong condition. Mrs. Hogans has suffered mental anguish in the past and will continue to suffer mental anguish in the future. Mrs. Hogans remembers how she was before her stroke and understands that not all of the tumor was removed.

Mrs. Hogans manifests depression and anxiety as a result of her condition.

Mrs. Hogans and her family have altered relationships as a result of her brain stem injury.

Mrs. Hogans suffers from physical disfigurement. She has disfiguring facial paralysis as well as disfiguring eating and drinking abilities and requirements. Her impaired and altered speech and her confinement to a wheelchair are also disfiguring.

Mrs. Hogans' conditions are permanent and likely to worsen as she ages.

Although Mrs. Hogans' physical and mental condition are permanent, she would benefit from participation in physical therapy, occupational therapy, and speech therapy offered by qualified therapists and quality programs.

3. Dr., Alex Willingham - board certified physical medicine and rehabilitation physician who provided a life care plan for Mrs. Hogans.

Mrs. Hogans suffered permanent injury, and will likely never work again as a result of her brain stem injury.

Mrs. Hogans' condition is as represented on plaintiffs' exhibits numbers 85 and 95, and both sides of her body are impaired. Mrs. Hogans will not likely walk again in a functional way.

Mrs. Hogans experiences physical pain and discomfort resulting from her brain stem injury. She is in a wheelchair for long periods of time and a certain degree of discomfort is consistent with her constant sitting and lack of normal movement abilities.

Mrs. Hogans has a full range of emotions, and she understanding her condition is lifelong. Mrs. Hogans has suffered mental anguish in the past and will continue to suffer mental anguish in the future.

Mrs. Hogans has memory of her normal self prior to the stroke and understands not all of the tumor was removed.

Mrs. Hogans and her family have altered relationships as a result of her brain stem injury. Prior to her injury, Mrs. Hogans was the matriarch of the family but now relies entirely on her family members to care for her needs and to serve as her daily heath care providers.

The Life Care Plan he prepared, as set forth in plaintiffs' exhibit 25, was carefully and accurately prepared after examining Mrs. Hogans, speaking with her and her family, evaluating their home and vehicle, and reviewing her medical records. The Plan is a culmination of his opinions concerning Mrs. Hogans' medical condition and all of the line items represented are reasonable and medically necessary to treat her medical condition as a result of the brain stem injury.

His Life Care Plan does not include costs for routine medical care that would have been needed regardless of the injury, loss of hearing, or for the treatment of diabetes, hypertension, or other preexisting conditions. His Life Care Plan will provide for Mrs. Hogans' future reasonable and medically necessary needs.

He takes issue with the care recommendations offered by Ms. Kuntz. Her care recommendation was not prepared in a reliable manner because Ms. Kuntz never spoke to or examined Mrs. Hogans, never interviewed Colonel Hogans, never interviewed any of Mrs. Hogans' other family members who have been caregivers for the previous three years, and never evaluated the home or vehicle. Due to these inadequacies, defendant's "care recommendations" cannot be called a Life Care Plan.

Mrs. Hogans' life expectancy is comparable to her statistical life expectancy. Her life expectancy was derived from the United States Government Life Expectancy Tables for black females which necessarily includes those with diabetes and hypertension.

The quality of care given to Mrs. Hogans will impact her life expectancy.

Mrs. Hogans wishes to remain at home and her entire family wants the same for her. His Life Care Plan and plan for attendant care will grant Mrs. Hogans and her family's wish.

His Life Care Plan includes therapy and attendant care for Mrs. Hogans. The United States Government has not provided therapy for her for almost two years and no future therapy has been planned by the Government. Additionally, the Government has never provided attendant care for her and there is no offer or plan by the United States Government to do so in the future.

Mrs. Hogans suffers from disfigurement—she has disfiguring facial paralysis as well as disfiguring eating and drinking abilities and requirements. Her speech is altered and the necessity of being in a wheelchair is also considered disfiguring.

Defendant's Experts:

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1. Kathleen R. Kuntz, MSN, RN, CRRN, CLCP, Cm—Ms. Kuntz has both a bachelor's degree and a master's degree in nursing. She is a registered nurse who is certified as a life care planner and a rehabilitation RN.

Her testimony is based not only on her education and career, but also upon the experience obtained in her personal life in caring for an elderly parent and a disabled child.

Her plan suggests approximately $1.5 million with discounting to present value. To fund this plan, $1,471,227.184 is needed. Her plan does not take into account what is available through TRICARE. Ms. Kuntz has provided for two scenarios: one in which Mrs. Hogans will remain in her home until her death and the other should Mrs. Hogans require institutionalization at age 65.

The most significant difference between Ms. Kuntz's plan and Dr. Willingham's Life Care Plan relates to delivery of attendant care services. Plaintiffs' plan sets forth a cost of approximately $2.5 million for attendant care and essential services while defendant's plan sets forth a cost of $1.1 million for attendant care and household cleaning.

Her plan utilizes an "Adult Day Program" in addition to 6 to 10 hours of attendant care at $13.50 per hour to deliver services for Mrs. Hogans. An attendant's duties would include assistance with personal care, meal preparation, shopping, errands, and light housekeeping. Her plan also allows for heavier household cleaning.

2. Dr. Stan Smith - a forensic economist.

His calculations and tables are contained in defendant's exhibit 60. His calculations assume a 7.42% discount rate and also assume an investment mix of 2/3 stock and 1/3 bonds.

His calculations provide for an earnings loss, past and future, of $195,707. A 1.2% discount rate was used for his earnings calculations, and in his opinion, Mrs. Hogans will suffer an earnings loss, past and future, in that amount.

The present value of plaintiffs' medical needs if $1.7 million for Dr. Willingham's Plan and $812,000 for Ms. Kuntz's Plan.

His methodology results in a present need of $1,730,193 to fund Dr. Willingham's Life Care Plan and $811,919 to fund option 1 of Ms.. Kuntz's Plan..

He did not double count household services like Dr. Huddle.